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COMMUNICATIONS

SOME NON-SURGICAL AIDS IN THE TREATMENT OF GLAUCOMA

BY

S. R. GIFFORD

OMAHA, NEBRASKA

FROM THE DEPARTMENT OF OPHTHALMOLOGY, UNIVERSITY OF NEBRASKA MEDICAL COLLEGE

THE first statement that ought to be made on this subject is a confession of my belief that nearly all cases of glaucoma will sooner or later require surgical intervention. It has been my experience to see many more bad results from postponing this intervention than from unsuccessful operations. Many ophthalmologists, for some of whom I have great respect, seem to have a horror of operating in simple glaucoma, and it is hoped that these notes will not offer any encouragement to an opinion which has, I believe, often sacrificed a patient's only prospect for retaining useful vision. There are, however, certain cases in which operation is particularly inadvisable, or in which it is desired to postpone it for a certain time and in these cases we may be grateful for any additions to our non-surgical armamentarium. In the November *Arch. of Ophthal.* for 1928 I summarized experiences culled from the literature concerning a number of therapeutic measures which have recently come into use, especially in Germany, and the exact references mentioned here may be found in that article. A number of these seemed of doubtful value, or with not enough clinical evidence to justify their general use. I thought to-night I would select for a

more detailed discussion a few of the newer methods with which substantial results have been obtained, and especially those of which I have had experience.

It goes without saying that the miotics, eserine and pilocarpine, should be tried in most cases before anything else is done. In an untreated case of simple glaucoma, these miotics will nearly always produce some fall in tension, and will often keep the tension normal for varying periods of time. They should be used often enough to keep the tension normal, if possible, or to ascertain how low it can be kept by their means, and I do not see how this can be done except by giving the patient a solution to use at home, for instance a solution of eserine one grain to the ounce, to use three or four times a day. The constant use of eserine may produce conjunctival irritation and in a few cases a conjunctivitis resembling that caused by atropine and in these cases pilocarpine is to be preferred, or the alternate use of the two drugs. On account of the occasional occurrence of synechiae after prolonged miosis, pupils which remain very small should be allowed to return to normal size occasionally, or even be aided by a drop of homatropine or cocaine, the patient being watched carefully afterwards for a return of raised tension.

The great danger of miotics is that of overconfidence in their effect, so that the vision and especially the fields as well as the tension, should be watched carefully during their use. The observation of Thiel, now amply confirmed by others, should be remembered, namely, that there is a characteristic daily curve of intraocular tension in glaucoma patients, tension usually but not always being highest on arising in the morning and lowest at night: hence, it should be recorded in the morning when possible, and when the field is failing, even if our tonometric findings are normal, it must be considered that a rise of tension probably exists at some time of the day or night, or at least a tension too high for the lamina cribrosa of the particular patient. In these cases, and the more common ones where miotics exert their effect only for a time and then fail to have any effect, something else must be done. It is understood that a careful general examination of the patient has been made, and measures taken to reduce blood pressure and clear up obvious pathological changes which might be a factor in the case. Frankly, in my own experience no very brilliant results have been obtained in glaucoma by constitutional corrective measures and I have seen only two general conditions which seem to have an undoubted aetiological relationship to glaucoma. These are the so-called vaso-neurotic diathesis, and the related condition of bronchial asthma. The vaso-neurotic diathesis includes persons of unstable vaso-motor mechanism and usually of unstable nervous system, as shown by tachycardia, flushing, angio-neurotic oedema and urticaria. Müller's observations by capillary micros-

copy of such patients have been extended by Scheerer and Parrisius to glaucoma patients, a large number of whom showed changes characteristic of the vaso-neurotic diathesis.

I have seen a number of cases of glaucoma in which marked rises of tension could be observed to follow attacks of asthma, or emotional shock, and believe the great therapeutic agent against these factors, aside from adrenalin in true bronchial asthma, is mental and physical rest. In the case of many patients, as soon as it is found that under the best possible control of general factors, and with the use of miotics, high tension persists, operation had best be undertaken at once.

Further delay may be considered advisable under some of the following conditions:

In very advanced age or feeble health; in secondary glaucoma where an access of inflammation is likely to follow any operation, especially in sympathetic ophthalmitis; in patients who have lost the sight of one eye—especially after an operation, with the field cut down close to the fixation point in the remaining eye; and lastly, in patients who refuse operation.

The cutting down of the field should not be ordinarily considered a contraindication to operation. While we know that following a successful filtering operation a loss of field may occur, which may involve the fixation-point, we also know that this same loss, followed by further loss, will occur in time if the tension remains high. When two precautions are observed, I believe these losses of field following operation may be made exceedingly rare. These are, to reduce the tension to as near normal as possible before operation so that the change in tension may not be too great, and to minimize the time during which the anterior chamber remains empty following operation, in which respect iridencleisis with running conjunctival suture presents advantages over trephining.

The best known of the newer methods of reducing tension is the use of adrenalin or glaukosan, which, although by no means original with Hamburger, has been popularised by his thirteen or more publications on the subject since 1923. Glaukosan is essentially nothing more than Hamburger's only too suggestive name for dextro-rotatory adrenalin, preferred to ordinary adrenalin because of the absence of systemic symptoms from rise of blood-pressure after its subconjunctival injection. While 1:1,000 solutions of adrenalin were ineffective by instillation, Hamburger found that a 2 per cent. solution of ordinary adrenalin when instilled several times during an hour, produced maximal mydriasis with a fall in tension as great as by subconjunctival injection of the 1:1,000 solution. He called this solution, to which was added an optically inactive substance produced in the manufacture of adrenalin, "linksglaukosan," or glaukosan. Because of its

strength, it is not to be used for injection, but for instillation only, the patient being placed in the supine position and two drops placed in the inner angle, after which the lids are held open for thirty seconds. This is repeated three to five times at fifteen minute intervals, according to the effect desired. I have made a two per cent. solution by dissolving one grain of adrenalin as provided in ampoules in 50 minims of distilled water, two drops of dilute hydrochloric acid being necessary to complete the solution. This gives similar results when used in the same manner as glaukosan but is not so stable. In most cases, the ordinary 1 : 1,000 adrenalin was used, first by subconjunctival injections of four to five minims, and later, by the method suggested by Gradle of inserting a cotton pledget soaked with the same amount under the upper lid and leaving it four minutes. Enough absorption occurs when it is used in this way so that the same effect on the pupil and tension occurs as by injection and without the systemic symptoms occasionally observed after injection. Only one or two instillations of two per cent. butyn are required as a preliminary, while with injections more thorough anaesthesia is required or pain will often be complained of. The effect of packs or injections on tension seems to be practically the same as that of glaukosan. Gredstedt, in a recent comparison of series where each was used, found the effect of adrenalin more marked and lasting. When the object is the dilatation of a contracted pupil in iritis, to break up adhesions, glaukosan or the two per cent. adrenalin solution present the advantage of being absorbed over the whole cornea and bulbar conjunctiva, so that a uniform dilatation is produced, with a better result in fresh synechiae than packs which affect most markedly the upper segment, or injections which affect the segment where the injection is made. I have only seen a few older synechiae which could be broken up by any of these methods, and have seen pupils dilate under them, only to return after twenty-four hours to their former size in spite of the subsequent use of atropine.

There is some difference of opinion among observers as to the type of case in which adrenalin is indicated. Most agree that it is ineffective in acute, absolute and haemorrhagic glaucoma. In secondary glaucoma and in iritis glaucomatosa, where it was enthusiastically recommended and where it would theoretically be of so much advantage by simultaneously dilating the pupil and lowering tension, a number of discouraging results have been reported. Vannas saw a rise of tension in three such cases and no effect in two others, and concludes that it is contraindicated in the presence of any inflammation. This agrees with my own experience, as I have seen two severe reactions in such cases with acute rise of tension. One was in a blind eye with a tension of sixty, containing a piece of copper, in which the patient wished to delay enucleation

several days. The pain following an adrenalin injection was so severe that he was glad to part with the eye the following day. The other case was a luetic patient with a haemorrhagic glaucoma in whom rise of tension with severe pain followed an adrenalin pack, lasting twenty-four hours, but resulting in a reduction of tension from forty to twenty-seven. These experiences have led me to discard the method in such cases, and reserve it for simple glaucoma, where it will serve, in a large percentage of cases, to reduce tension before operation, or when used with miotics, to allow postponement of operation for long periods.

Out of about one hundred treatments, the records of fifty are complete enough to be of any value. In two-thirds of these, packs were used, in the rest, subconjunctival injections. In fifteen cases, the treatment failed to produce an appreciable decrease of tension. These failures were in cases of iritis glaucomatosa 2, inflammatory glaucoma 2, absolute 1, secondary 1, acute 1, and simple 9 cases. In all the others, most being cases of simple glaucoma, a marked fall of tension occurred, the average decrease being 10 mm., while individual falls of 20, 23, and 24 mm. were recorded. In all but three of these, tension reached normal or below 25 mm. Schiötz. The tension was not taken often enough to determine the average duration of decreased tension, but in thirteen it remained normal one week or more and in five one month or more. Miotics had been tried in all cases before the treatment and were continued afterwards. Some cases left observation after one or more packs with tension still normal, while in a number of others later injections were less effective and operation was performed. Four cases have been followed for eight to twelve months, all having lost the sight of one eye elsewhere on other forms of treatment, and in these four vision has remained the same as at first, and tension has remained normal on miotics with an occasional pack, except in one case in which only a week ago the pack was finally ineffective and iridencleisis was performed on the only seeing eye. Certain cases responded much better to this treatment than others, and I have a distinct impression that it is especially apt to be effective in those of the vaso-neurotic type, who are subjects of vagotonia and hence would theoretically be especially responsive to the sympathetic stimulant adrenalin. I mention this with hesitation, as I know of no subject on which such floods of ink have been spilled to so little purpose as this one of vagotonia.

In simple glaucoma, the only complication seen was an acute rise of tension occurring in two cases which were reported in the *Amer. Jl. of Ophthal.* (1928, 11, p. 628). While no permanent ill effects resulted in these cases, they were especially dramatic as involving in each case the only seeing eye. In one, the tension remained high following paracentesis, but subsided during twelve

hours, then remained normal for two weeks under eserine, when it rose to 44 mm. (Schiötz) and iridencleisis was performed. In the other, it subsided after three hours and has remained about 14 mm. (Schiötz) for eight months up to the present time. These attacks, while rare, have been reported by others in simple glaucoma, and in one case, that of Böhm (*Arch. of Ophthal.*, 1928, p. 574), resulted in permanent reduction of vision to hand movements in one eye. In this case no miotics were used for two days afterwards and operation was delayed five days, and the disastrous result could probably have been prevented by miotics or paracentesis. It is on account of these occasional attacks, however, that I consider inadvisable the practice of Thiel, who gives patients an adrenalin ointment to use at home. These cases should be watched for some time after treatment. Eserine should be given twice at five minute intervals before treatment, and repeated once every twenty minutes in the first hour afterwards, and every half-hour in the next two hours, or often enough to keep the pupil small. This use of miotics does not lessen the effect of the drug on tension.

Before leaving the subject of adrenalin and glaukosan, aminoglaukosan must be mentioned, if only to clear up the confusion due to its name. This is Hamburger's name for a ten per cent. solution of histamin, a protein split product derived from ergot. It has the opposite effect of glaukosan, one drop instilled in the sac being sufficient to contract a pupil dilated by atropine to pin-point size in seven to fifteen minutes, according to Hamburger. It is recommended by him in acute glaucoma, the resulting miosis having reduced tension in a limited number of cases. Others, however, have reported failures and the accompanying chemosis of the conjunctiva has necessitated delaying a needed operation in several cases. I have seen such a severe inflammation follow its use in one case that I have not been encouraged to try it further.

Another procedure which will reduce tension in acute glaucoma, if only temporarily, and which has the virtue of being without danger, is the systemic use of hypertonic solutions. This is done with the idea of making the blood plasma hypertonic, causing absorption of fluids from all the tissues, including the eye, into the blood stream. That this actually occurs is attested by the results of Cantonnet, Hertel, Weekers and others. 35 to 50 c.c. of 30 per cent. or 100 to 150 c.c. of 10 per cent. sodium chloride are injected intravenously, the stronger solution producing the more rapid effect. Duke-Elder reports reduction of tension from 95 to 38 mm. by this method in one case and from 58 to 20 in another. He summarizes the indications for the method as follows:

"1. In acute glaucoma, in conjunction with miotics, before operation.

"2. To reduce high tension before any operation. Besides lessening the danger of vitreous loss and haemorrhage, reduction of tension allows the absorption of local anaesthetics, which are ineffective in eyes with high tension.

"3. In iritis glaucomatosa, before using atropine.

"4. To facilitate ophthalmoscopic examination where the cornea is steamy from increased tension.

"5. Is of possible value in any intraocular inflammation by withdrawing fluids from the eye, these being replaced by fresh fluids from the blood stream, bearing increased anti-bodies or drugs which have been given. It would act theoretically like a paracentesis."

As no traumatism is inflicted on the eye itself there is no danger in using this method in any form of glaucoma, and it does not increase the technical difficulties of operation, as amino-glaukosan may do.

Solutions of 30 per cent. glucose have been given by Sansum to produce the same effect on the blood, but large amounts must be given (300 to 500 c.c.). Magnesium sulphate in large doses by the mouth may also extract enough water from the blood by way of the intestinal tract to lower the intraocular tension and is used by neurological surgeons for the same effect on intracranial tension. The effect of these solutions by the mouth, while less rapid than when the intravenous route is used, is more prolonged.

I have had personal experience with this method (*i.e.*, the use of hypertonic solutions) in only three cases, having been deterred from using it by the time required for preparation of solutions and apparatus, and by the circumstances under which operations for acute glaucoma must often be done, but I believe the complications from operation, especially haemorrhage, would be less frequent if it were more often used, and that a few eyes might be brought under the influence of miotics so as to render operation unnecessary.

Of a number of drugs less well known in America than adrenalin, I have wished to discuss here gynergen or ergotamine, because the reports concerning it, while not as yet very numerous, seem to agree fairly well on the results of its use, and because I have had some experience with it. This is a derivative of ergot, containing the active principle used in obstetrics. It is used subcutaneously in a dosage of 1/250 grain or by mouth 1/60 to 1/30 grain. Besides causing contraction of the uterus, it raises the blood pressure. It depresses the sympathetic nerve-endings and its use in glaucoma is based on the idea that hypertension is due to hyperactivity of the sympathetic system, and that such a drug would decrease the permeability of the vessels.

Thiel reports on 90 cases of glaucoma in which ergotamine was used by subcutaneous injection of 1/4 mg. or by oral administration of one to three tablets of one mg., doses by each method being repeated three times a day. Rest for one hour after treatment is advised to avoid systemic symptoms observed in a few cases. A summary of results in these 90 cases is not given but a few illustrative cases show that in simple glaucoma the tension could be kept normal by oral administration of ergotamine for several months, and that after the first three to four weeks miotics which had been ineffective, were so much more effective that the ergotamine could be discontiuued. In one of these cases the tension sank from 70 to 24 mm. after one subcutaneous injection. In iritis glaucomatosa and some cases of secondary glaucoma where iritis was present, good results were obtained, and in one of these an acute attack of glaucoma was aborted by one injection. No effect was secured in absolute glaucoma. In inflammatory glaucoma a fall from 72 to 24 was observed in one case. Römer and Krebs observed reductions of tension in glaucoma produced by ergotamine, but not so great as would be produced by adrenalin. Heim confirmed Thiel's observations in a series of cases and also found that after using fairly large doses for a variable time much smaller amounts were effective in keeping the tension normal, when combined with the use of miotics. The advantages of any method of treatment that can be given systematically, without traumatism to the eye, are obvious in certain cases of glaucoma.

I have used ergotamine in eleven cases of glaucoma in which operation was for some reason contraindicated. Five were cases of glaucoma after cataract operation, one of which was a traumatic cataract with anterior synechiae. Three were of glaucoma in sympathetic ophthalmitis, two were of simple glaucoma and one of absolute glaucoma. In most cases one-half c.c. of the solution was injected subcutaneously twice a day, in a few it was given by the mouth, from one to three pills three times a day. In two of the cases of glaucoma following cataract operation, the results were disappointing, tension being reduced only by 2 to 4 mm. (Schiötz) so that other means had to be employed. This was also true of the case of traumatic cataract. In one case, however, tension fell from 30 to 18 three hours after one injection and was kept down to 24 by oral administration, with an occasional injection, for three months, being 24 at the last examination, when vision with correction was 20/40 and no ergotamine had been used for several weeks. In another case tension fell from 32 to 24 and later from 37 to 17 mm. following injection, but these could not be continued on account of nausea.

In one case of simple glaucoma, where eserine and adrenalin packs had finally lost their effect, not enough effect was produced

by ergotamine to avoid operation. In the other case a fall of tension from 37 to 11 mm. occurred five hours after one injection but this was an atypical case whose tension varied greatly owing to other factors. In the case of absolute glaucoma, tension fell from 70 to 60 mm. following one injection and six oral doses. In one case of sympathetic ophthalmitis with iris bombé in a child of six, four oral doses caused a fall of tension from 44 to 37 in twenty-four hours, after which the case was not seen. In a second case which could be watched in the hospital, a regular effect of the drug was seen time after time, decreases of tension from 40 to 25 mm., and 35 to 20 mm. being recorded repeatedly. A slighter effect on tension was produced when the drug was given by mouth. In this case the use of gynergen was of the greatest value, as operation could be postponed for three weeks until much more favourable conditions were present and a post-operative rise of tension was controlled. In a third case tension was reduced from 32 to 19 mm. after one injection but three days later was 37 mm. and this time the drug was ineffective.

It will be seen that, as with adrenalin, cases vary greatly in their reaction to this drug, and that in a large proportion, its effect was not sufficient to avoid operation. This might be expected in the cases following cataract, where purely mechanical factors of drainage are apparently concerned, and still it was in one of these that the most brilliant result was obtained. If it were only for this case and the two cases of sympathetic ophthalmitis, I would feel that the time spent in experimenting with ergotamine was not wasted. I am sorry not to be able to report more experiences in simple glaucoma, which is where one would hope for the best results. One thing can be said for the drug, that it has caused no ill effects in my experience. One patient complained of such headache and discomfort following its use that it had to be discontinued, and one had diarrhoea, but the others noticed no subjective symptoms. The purpose of these notes is to show that it will decrease tension in certain cases, and that a trial of its effects is never harmful, unless too much time is wasted before a decision is arrived at.

The same may be said of several other drugs, pituitrin, tyramine and barium, from the reports of others, but since these reports were not so convincing as those concerning ergotamine, I have thought it best not to discuss them here. Brief notes concerning them will be found in the November *Arch. of Ophthal.*, 1928. With another method of treatment, the administration of calcium as calcium chloride internally, or as afenil intravenously, I have had no experience but believe that the method is worth mentioning. It rests upon the fact that calcium decreases the permeability of the capillaries, and the results of Weekers, Gowland, Alt, Kleiber and

von Hofe indicate a definite value in the method, which can be used with miotics. von Hofe found that calcium increased markedly the effect of adrenalin given subconjunctivally.

Summary

1. Adrenalin or glaukosan is of value especially in simple glaucoma, and by its means tension may be kept normal for considerable periods in certain cases.
2. Miotics should be used to prevent acute rise of tension when using adrenalin and to prolong its effect afterwards.
3. Amino-glaukosan presents the danger of increasing inflammation, and fails to reduce tension in many cases.
4. Hypertonic solutions can be depended upon to reduce tension before operation in acute glaucoma, and are without danger to the eye.
5. Ergotamine will reduce tension appreciably, it may be of much value in a small percentage of cases, and may be tried safely in any case.
6. All methods of treatment demand careful watch of the vision, fields and tension, so that progress of the disease may be noted early enough for effective surgical intervention.

A CASE OF MONOCULAR BLINDNESS OF ELECTRICAL ORIGIN*

BY

J. N. ROY

ASSOCIATE PROFESSOR AT THE UNIVERSITY OF MONTREAL
PHYSICIAN TO THE NOTRE-DAME HOSPITAL
LAUREATE OF THE ACADEMY OF MEDICINE OF PARIS

To my knowledge medical literature relates the observation of seven cases where the two eyes have been lost by the dazzling effect of electric light, after having been exposed to the intense luminous rays caused by lightning, by reverberation of the sun, and finally by a short circuit. As the patient—the subject of this work—has become blind of one eye only, after having been submitted, at a short distance, to the effects of electricity and of melting metal, I believe it interesting to publish his history, and will profit by this circumstance to recall the most recent theories on the injurious action of intense light in relation to the visual organ.

Case report.—On May 25, 1927, Mr. A. B., aged 37 years, labourer in a foundry, received the order to carry a bar of steel into an electrical soldering room. Entering these premises for

* Read before the *10th Congrès des Médecins de langue française de l'Amérique du Nord*, Quebec, September, 1928.

the first time, he was keenly impressed, especially by the description of the workings of the machinery by his companions. Also, at a distance of four to five feet from the luminous source, he looked with persistence, during a period of about eight minutes without any protective glasses, at the rays emitted by a mass of metal in a state of incandescence and disintegration under the action of a strong electrical current. During the hours which followed a double ophthalmia declared itself, and a few days later he noticed a trouble and a weakness in the vision of the right eye. The palpebral and conjunctival objective symptoms of the two eyes disappeared rapidly, as they always do with the ordinary therapeutic treatment, but the visual power of the right eye diminished gradually. At the time of my examination, on January 15, 1928, I observed a cicatricial lesion of the retina, near the disc, on the temporal side, a lesion of the macula lutea, a narrowing of volume of the retinal arteries, and finally a generalised discolouration of the papilla, accompanied by a definite optic atrophy of this eye, which had reduced the vision to the point of luminous perception. The patient, being seen for the first time, it is impossible for me to describe the neuro-retinal alterations which have successively followed since the time of the accident. However, I have been able to know from the oculists who have observed him during the few months which have followed the dazzling, that on June 12, that is to say 18 days after his exposure to the luminous rays, the vision of the right eye was 1/10; on July 27 the vision was still 1/10; on October 7 a second oculist found it unchanged, and since that date the visual acuity had gradually diminished till it disappeared almost entirely.

As to the left eye, I have found that the condition was normal from all points of view.

Let us now rapidly see how light acts upon the eye. The action of light upon the visual organ may be shown under different aspects, but what is interesting for us to note is the effect that it produces on the retina. Daylight, as we know, is decomposed by the prism to a spectrum of which one part is visible; the two extremities, the infra-red and the ultra-violet, do not cause, in ordinary circumstances, a sensation of vision in the human eye.

We will then put aside the action of the ultra-violet and infra-red rays. Indeed, the first are nearly all absorbed by the middle parts of the eye which protect the retina; this is what seems to be the result of the numerous researches made on this question. The second would have especially some calorific properties more characteristic to it, but whose intensity upon the retina seems to be negligible. There remains, between these two extremities of the spectrum, the visible light which has not yet been absorbed by the middle parts, and which brings to the retina a luminous

energy which this, after absorption, must transform into chemical, caloric and electrical effects, which will determine or accompany the visual sensation.

Whether light may be constituted according to the quantum theory, or by undulatory radiations, or by the two at the same time, in every hypothesis, it is necessary that it should be absorbed before acting and producing photo-chemical, photo-caloric, photo-electrical and photo-sensorial modifications on the retina.

These varied transformations of energy are not made without chemical or biological modifications of the retina, which are dependent on deep electronic modifications. Between certain limits, these modifications are reversible, beyond which they are destructive and permanent; the absorption which arranges them depends on the nature of the absorbent centres, on the wave length, and on the intensity of the incident radiations. Thus it is not astonishing that the radiations of a very intense visible light might produce serious and irreparable lesions of the retina by electronic modifications which give but very little heat.

The deeper study of the direct action of light upon the retina and, consecutively, upon the optic nerve, will help us later on to understand better some lesions and some biological phenomena that we are accustomed to associate with the effect of heat.

All that I have stated in relation to daylight naturally refers also to electrical or other lights. Consequently, it is easy to understand that if a visible luminous source of a weak or medium intensity does not produce any harmful action upon the retina, in other circumstances a powerful light can cause, on the fragile elements of this membrane, transient or permanent lesions which may sometimes terminate by complete atrophy of the optic nerve.

We know that electrical ophthalmia can produce, upon the skin of the face and of the eyelids, and on the anterior segment of the eye, especially by the effect of invisible ultra-violet chemical rays, and of invisible infra-red caloric rays, a series of changes including epidermitis, palpebral oedema, conjunctivitis, keratitis and iritis. I do not believe it necessary to insist more on this point, because these observations are admitted by all authors. However, I will recall that opinions are rather divided as to cataract of an actinic origin. A certain number of experimenters consider that the ultra-violet rays have the property of rendering the crystalline lens fluorescent, and that these invisible rays of a short wave length transform themselves into visible rays of a greater wave length. In admitting this hypothesis, it is easy to figure to ourselves that such a transformation of energy has the probable effect of modifying the crystalline body where the reaction is produced, and consequently that cataract is possible. Other observers are of opinion that it is the invisible infra-red radiations which are

responsible, and that the lental opacity is dependent on these latter rays. It is needless to add that numerous laboratory experiments have been made upon animals before arriving at such assertions. And finally, a certain number of authors believe that neither the ultra-violet nor the infra-red rays can produce a cataract. However, it is a clinical fact of current observation—which I have myself noticed during my numerous travels in tropical countries, especially in Africa, in Oceania, in Central and South America, and particularly in Asia—that cataract is much more frequent in these latitudes than in countries where the sun is less powerful. Though, I believe that the very strong heat of these regions tends to modify the ocular nutrition, and that this phenomenon must be considered in discussing the aetiology of cataract. In the same order of ideas, I ask myself moreover, if the diet of the natives of these torrid zones, composed especially of fruits, vegetables and corn, must not also be considered.

As to the lesions of the deep layers of the eye observed following electrical dazzling, the retina is the membrane first affected. The macula lutea is particularly vulnerable to powerful luminous rays, and its alteration produces a central transient or permanent scotoma. The retina loses its transparency, and presents the aspect of a more or less oedematous cloud, surrounding the optic nerve and the large blood vessels. The papilla may remain normal; however, in certain circumstances it becomes inflamed, or simply "*floue*," and its borders appear lightly stippled. The visual acuity is diminished by alteration of the central vision, and sometimes the visual field is narrowed. In the majority of cases all these phenomena disappeared after a certain time, and the dazzling did not leave any trouble of the eye. In others, on the contrary, the papillo-macular bundle becomes atrophied, and this lesion produces a permanent central scotoma. And finally, when the light perceived has been intensive, it may result in cicatricial islets of the retina, a diminution of volume of its blood vessels, and finally a discolouration and an atrophy of the optic nerve, ending in blindness. Though these facts are not sufficiently discussed in the treatises of ophthalmology they are well known, especially by those oculists who have observed patients seriously affected by electrical ophthalmia.

I should like to recall the few rare cases published in the medical literature where amaurosis has ensued.

Brière gives the history of a young girl aged 11 years who, after having walked during a violent storm, on a road illuminated by flashes of lightning, presented ophthalmoscopical symptoms of a double neuro-retinitis, and finished by losing her eyesight.

Römer relates the case of a boy, aged 12½ years, who was overthrown by lightning which struck a tree at a distance of a

few paces from him. The final result of the shock and the dazzling was a double papillary atrophy.

Knies also mentions a case of optic atrophy in a child, aged 10 years, following electrical ophthalmia.

Brandenburg publishes the case of a patient who had a neuro-retinitis followed by an optic atrophy after having worked outside all day long under an ardent sun.

And finally, Terrien, who has given special attention to this question, after having observed about fifty subjects during the construction of the Metropolitan Underground Railway of Paris, has seen atrophy of the optic nerve following electrical dazzling on three occasions.

In my patient, it is interesting to note that the deep layers of his left eye had not been hurt at the time of the accident, and that the vision of this eye was normal. However, it is natural to suppose and admit that when he was looking at the very strong light, a reflex movement had caused his head to turn towards the left side, and thus placed, the corresponding visual angle of this side presented less surface to the effects of the injurious visible rays upon the left retina, the pupil being protected by the nose.

In the present case, must we believe that the lesions of the retina, and consecutively the atrophy of the right optic nerve, have been produced by the electrical source itself, or by the visual light from the molten metal, or by these two kinds of radiations? It is rational to admit that all the incandescent particles had contributed to the ultimate effect: those that the electric arc had detached, as well as those which had remained adherent to the solid; but each one according to its temperature and its characteristic radiations. It is probable also that the rays had fallen slightly obliquely upon the face, and that the active luminous surface had not been very extensive, since the left eye had been protected by the nasal pyramid in a slight deviation of the head. To sum up, this case of monocular blindness of electrical origin—unique in medical literature—is very interesting.

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HYPOPYON ULCER OF THE CORNEA DUE TO GLENOSPORA GRAPHII

BY

LT.-COL. R. E. WRIGHT

PROFESSOR OF OPHTHALMOLOGY, MEDICAL COLLEGE, AND SUPERINTENDENT, GOVERNMENT OPHTHALMIC HOSPITAL, MADRAS

N., a Hindu female, aged 38 years, presented herself on August 14, 1928, with a hypopyon ulcer of the cornea. The lesion was round, about 3 mm. in diameter, situated in the centre of the inferior quadrant. An observer at first sight might have accepted the case as an ordinary ulcer with hypopyon, although there was a difference, which, without going into tedious detail, might be summed up by saying that the whole picture was more indolent than the usual hypopyon ulcer, using the word in its ordinary surgical sense. There was one feature, however, which could not have escaped the eye of an alert ophthalmologist: the floor of the ulcer was filled in by a tough slough, so that when one explored it with a suitably pointed piece of match wood soaked in pure carbolic, it was possible to demonstrate a slightly undermined edge, a gutter of demarcation, and a greyish yellow, and tightly adherent, central mass. After preliminary surgical cleaning of the conjunctival sac and superficial sterilisation of the slough, the latter was firmly seized in suitable forceps and removed with some difficulty. It was broken up in a sterilised Petri dish and cultures made at once on Sabouraud's medium. Under the microscope a dense mass showing an indistinct mycelium was observed, about which there was

nothing informative to be made out. Some pure cultures of the mould were obtained and sent for identification to Lt.-Col. H. W. Acton, I.M.S., Professor of Pathology and Bacteriology, and Director, School of Tropical Medicine and Hygiene, Calcutta, who was working on the pathogenic fungi. His report is given below.

The history of the case is interesting. The woman was exceptionally intelligent and independent, and apparently managed her own fields. Whilst working in the paddy (rice) crops about six weeks before, she stooped and her cornea impinged on a piece of stubble (rice or possibly other stalk). At the time she was treated in her own house, it is said, by tobacco juice and opium water. It was very painful, which is not to be wondered at, and she sought the advice of a local mission dispensary doctor who treated her for some time. The condition got worse and being anxious for further advice, she asked her son to bring her to Madras. This fixes the nature of the implantation and the duration of development.

Treatment and Duration of the Condition under Treatment

After removal of the slough the floor of the ulcer was cauterised with pure carbolic. Thereafter atropine was regularly employed, and the ulcer touched, about twice weekly, with tincture of iodine. Potassium iodide was exhibited in 30 grain doses per diem by the mouth, and iodine given intravenously in dilute solution, in 0.25 grain doses twice weekly for four weeks. Locally magnesium sulphate irrigations, hot fomentations, yellow oxide of mercury and dionine were employed. The hypopyon rapidly disappeared, but the ulcer was slow to heal and the cicatricial tissue did not harden into a safe scar till three weeks after admission. She was discharged on September 9, 1928, with 6/18 vision in that eye.

Bacteriological Report

The fungus belongs to the Aleuriosporineae of the genus Glenospora described by Berkeley and Curtis, 1876, as having the following characters. Aleuriosporineae without conidiophores, with simple or appendiculate spores and pale and dark hyphae. The Aleuriospores become dark and are situated acropleurogenously, *i.e.*, situated at the end on dark or light hyphae, but are small, being generally 6—4μ in diameter and rarely 11—5μ.

In Pollacci and Nannizzi's paper on " Pathogenic mycetoma of men and animals " it is mentioned that Morax found Glenospora graphii in a case of mycosis of the cornea in 1910. Pinoy determined his cultures to be Verticillium graphii, Harz et Bezold, a species which Vuillemin afterwards assigned to the Glenospora. The authors state that probably several other cases announced by the generic name of mycosis of the eye, without determining the mycelium, may have been caused by the Glenospora graphii.

They note that Bianchini, during inquiries made in the Botanical Institute at Siena, found this species on the remains of exhumed corpses and that the patient described by Morax dealt with hides.

Coloured illustrations representing the growth as it appeared on various media, and the efflorescence, are not sent herewith as they would probably cost much to reproduce. Col. Acton has given me a copy of the original plates for the museum of the Government Ophthalmic Hospital, Madras, and I propose to send a duplicate to the Ophthalmological Department, London School of Tropical Medicine and Hygiene.

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THE OPHTHALMOSCOPIC PICTURE OF NAEVUS (MELANOMA) CHOROIDEAE

BY

KENNETH B. JOHNSTON

MONTREAL

THE object of this paper is to report three cases of naevi of the choroid, seen clinically, and to offer an explanation of their varying appearances.

I wish, here, to thank Dr. A. Fuchs for his interest as well as for his permission to publish the following three clinical cases which are from Professor Meller's Clinic in Vienna. Acknowledgment is also due to Prof. E. and Dr. A. Fuchs for their kindness in allowing me to study their pathological slides from nine cases of naevi.

The choroidal naevus is an important anomaly of pigmentation of the choroid. It has been extensively studied histologically and reported on by numerous authors, but few clinical reports can be found in the literature. Foster Moore[1] describes four cases of melanoma of the choroid, one of which he examined histologically. He publishes clinical drawings and gives an extensive bibliography of previously reported cases. He says, " In size they varied from about one half the area of the optic disc to about four times its area. They were roughly circular, or oval in outline. The edges were everywhere quite definite without being quite hard and sharp. There was no fading off into the surrounding fundus, nor was there

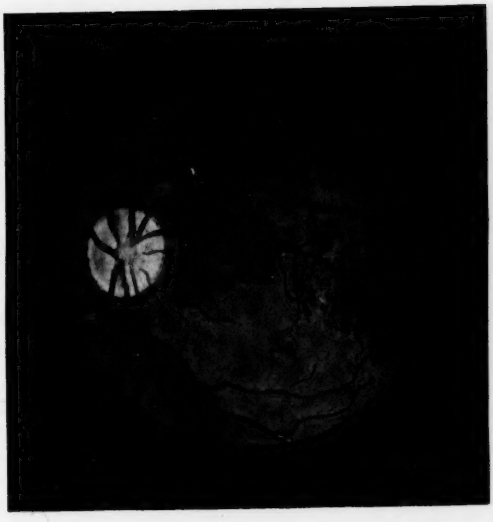

FIG. 1.

any fringe or evidence of pigmentary disturbance at the edge. They were of quite homogeneous appearance and the choroidal pattern though plainly seen around (in two cases) was not seen over the area of the growths. In colour they were exactly that of ' blue ointment ' differing only in their density. All were single and close to the optic papilla the one farthest away being distant about two discs breadth."

In his histological description he states that the area is an intensely black circular spot entirely limited to the choroid, the overlying retinal pigment epithelium being quite uninvolved, and the choriocapillaris for the most part quite clear. In a later paper[2] he publishes a second case again studied both clinically and histologically confirming these findings. He concludes that these growths are benign in character and probably congenital in origin.

In a discussion on melanomata[3], Foster Moore reports that in an examination of one of his cases five years and two months later, the growth was quite unaltered in appearance. Another case three years later showed no change.

Usher[4] also at this discussion reports a similar case examined both clinically and histologically. This naevus appeared a dark grey, circular in outline, and edge not sharply defined. It was situated two discs diameters from the disc and was two-fifths of its size. It was crossed by the inferior temporal artery which was not appreciably raised. Microscopically this area showed a collection of pigmented cells that resembled normal choroidal chromatophores. These cells are brown, contain granular pigment but not in sufficient quantity completely to obscure the nuclei. The choriocapillaris is not invaded by these cells but the whole width of the choroid is thickened at the site of the melanoma.

E. Fuchs[5] describes a case seen clinically, and publishes a series of excellent microphotographs of naevi from his collection including the section of the naevus from which the histological picture in this paper is reproduced.

A. Fuchs[6] in his Atlas illustrates and describes a naevus.

Magitot[7] reports a case in which the naevus had been missed ophthalmoscopically due to oedema of the nerve head but the histological sections show a typical naevus of the choroid which he reports in detail.

Some other observers describing this anomaly are Wolfrum,[8] Dimmer,[9] and Segalowitz.[10]

I wish to describe three cases of naevi because these show different clinical pictures according to the three possibilities of pigment distribution. These three well known types are the brownish-red fundus, the tessellated, and the albinotic fundus. These three types are produced by variations in the amount and colour of the pigment in the retinal pigment epithelium and the chromatophores of the choroid.

Cases 1 and 3 are chance findings in patients who came for refraction, and have no sign of any disease present. Case 2 is a woman, aged 66 years, with senile cataract. Two weeks following cataract extraction, vision was 6/8 and J.1.

Case 1 (Fig. 1). The naevus lies to the outer side of the macular region. It is a slightly elevated, round plaque, slate-grey in colour with an indistinctly defined margin. In size it is a little larger than the disc. The fundus is of a brownish-red colour; no details of the choroid are visible. The retinal vessels and disc are normal.

In this type of fundus the pigment epithelium contains a dense amount of dark pigment which completely obscures all the underlying choroidal details. For this reason one would expect a blurred outline of the naevus which, here, is actually the case. This fundus is the most common in appearance and the naevi, as a rule, are indistinctly outlined.

Case 2. In this case the naevus is situated above the macula, is slightly elevated and disc shaped. The size is approximately that of the optic disc, the colour is a slate-grey but the outline is quite definite. The fundus is tessellated, the choroidal vessels being seen everywhere as reddish bands except in the area of the naevus where they disappear. The disc and retinal vessels are normal. This naevus differs from Case 1, in having a much sharper outline. The pigment epithelium in this eye is poorly pigmented. Thus not only are the choroidal details more visible but also the outline of the naevus. The appearance of such a naevus is much more rare than that of the first case.

Case 3. This naevus is found situated in the periphery of the fundus. The magnification is greater than in Fig. 1. Actually, it is slightly larger than the disc, of a slate-grey colour and with a sharp outline. Retinal vessels are seen running horizontally across the drawing as thin purplish-red lines, the upper one overlying the border of the naevus. The choroidal vessels, larger and of a brick-red colour, are seen sweeping from below upwards and across the naevus, like an open fan. The naevus is thus seen, crossed by both retinal and choroidal vessels, the latter in fact, appearing to groove it. Between the choroidal vessels, the intervascular spaces are unpigmented and filled by fine branching vessels, probably from the praecapillary layer. This is a case of naevus in an albinotic fundus where the outline of the naevus and choroidal vessels are clearly visible on account of the poor pigmentation of the pigment epithelium. It is especially interesting as it demonstrates clinically that the naevus lies in the outer layers of the choroid, since the choroidal vessels of the medium-sized layer (Sattler) are seen crossing it.

In the three cases there is seen no sign of any proliferation or change in the overlying pigment epithelium.

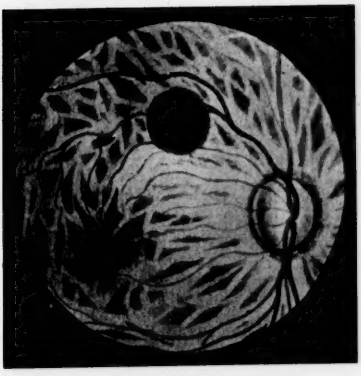

FIG. 2.

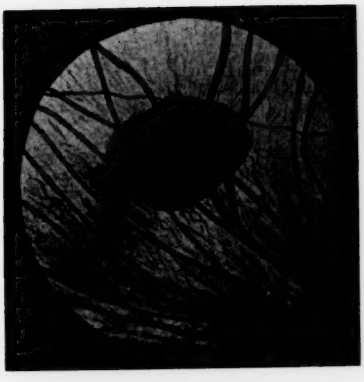

FIG. 3.

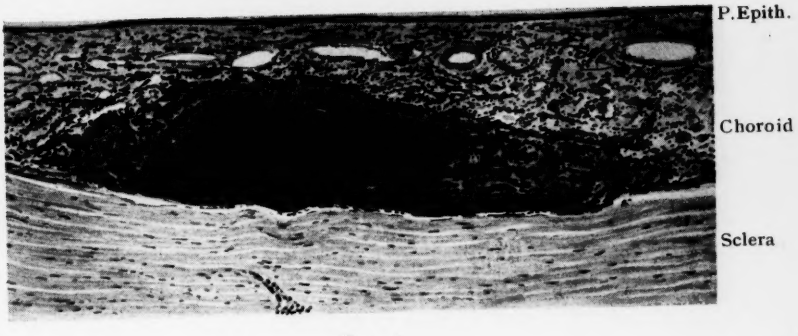

FIG. 4.

In making a histological examination of naevi I do not wish to go into the detail of the histological structure but rather to enumerate the histological points which have a bearing on the clinical picture. Four of these cases were published by E. Fuchs in *Arch. f. Ophthal.*, a fifth case appears in the *Atlas of Histopathology* of A. Fuchs, and four cases are new.

The histological drawing (Fig. 2) shows a naevus of the choroid lying in the external layers only. The outline of the naevus is more definite to the left than to the right. The pigment epithelium above is intact with a moderate amount of pigmentation. The choriocapillaris and several larger vessels of the middle layer are not invaded by the naevus cells. The cells themselves are densely pigmented although in the remainder of the choroid the chromatophores have only a very moderate pigmentation.

The accompanying chart shows the relation between the pigmentation in the pigment epithelium of the choroid and naevus. These nine cases were only histologically studied and had not been previously seen with the ophthalmoscope.

In all the cases but one the naevus lies in the neighbourhood of the disc. The width of the choroid in six was increased. I do not think, however, that the amount of the thickening would be enough to be measured by the ophthalmoscope. Foster Moore was doubtful of the measurement of 2 D. in one of his cases; Usher was unable to measure any swelling in his case.

In all the cases the naevi occupied the outer layers of the choroid leaving the choriocapillaris free. In three cases even Sattler's layer was partly free from naevi cells.

In all but one case I have noted the outline as definite. This is only a comparative term referable to the remainder of the choroidal pigmentation. The section of the naevi under the low power of the microscope has more of a rectangular than a pyramidal outline as might be expected from the feathered outline of the clinical picture. This blurring effect is due more probably to varying amounts of pigment in the overlying pigment epithelium. In five of the cases the pigment epithelium is noted as poor. One cannot be sure, however, if the amount of pigment that seems poor in a section is sufficient or not to hide the choroidal details in a clinical examination. The choroidal pigmentation was moderate in six cases, actually poor in two, and only dense in one case. On the other hand the pigment in the naevi cells was very dense in seven and moderate in two cases.

It is surprising that the clinical appearance is a slate-grey in spite of the dense black pigmentation of the naevus. Apparently only proliferation of the pigment epithelium can make a really black-looking pigmentation of the fundus, while the accumulations of the chromatophores give the effect of greyish discs.

Conclusions

1. The colour of a true naevus clinically is always a uniform slate grey.
2. The outline varies from being feathery to sharp, depending on the amount of involvement of the inner layers of the choroid, and the amount and colour of the pigment in the pigment epithelium.
3. The elevation is rarely sufficient to be measured clinically, although the choroid is usually thickened.
4. The size and shape do not alter over long periods (five years) of observation. (Foster Moore).
5. The retinal pigment epithelium is not involved in the naevus.

Since sarcoma of the choroid tends usually to grow inwards, we may expect to see some sign of pigmentary disturbance of the pigment epithelium over the naevus as a very early sign of a beginning malignancy from such a source.

No.	Location.	Width of choroid.	Outline of naevus.	Vessels NOT involved by naevus.	Pigment of pig. epithel.	Pigment of choroid.	Pigment of naevus cells.
1	Near disc	Not thickened	Definite	Chorio-capillaris	Chorio-retinitic changes	Medium	Dense
2	,, ,,	Thickened	,,	Choriocap. and middle layer	Medium	Poor	,,
3	,, ,,	,,	,,	Choriocap.	,,	Medium	,,
4	5 mm. from disc	Much thickened	,,	,,	Poor	Dense	,,
5	Near disc	Thickened	,,	,,	,,	Medium	Medium
6	Near Ora S.	Not thickened	Indistinct	,,	Chorio-retinitic changes	,,	Dense
7	Near disc	Thickened	Definite	Choriocap. compressed	Poor	,,	,,
8	5 mm. from disc	Slightly thickened	,,	Choriocap. and some middle vessels	Medium	,,	,,
9	Near disc	Not thickened	,,	,,	Poor	Poor	Medium

Nos. 1—3 Reported by E. Fuchs.
,, 4 ,, ,, Salzmann.
,, 5 ,, ,, A. Fuchs (Atlas).
,, 6 Atypical case.
,, 7—9 Not previously reported.

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EXOPHTHALMOS AND MIXED ASTIGMATISM CAUSED BY LARGE ETHMOID MUCOCELE

BY

E. A. SEALE, M.D.

GRAHAMSTOWN, S. AFRICA.

PATIENT, a man aged 20 years, consulted me on February 15, 1928, for a swelling about his left eye. He stated it began about four years previously and had gradually increased to its present size. Headaches were frequent and severe—the pain going through to back of head, and vision was affected. Some epiphora was present. There was a trifling redness over the swelling which occupied the inner orbital angle. On palpation it felt hard and firm; pressure gave a crackling which was both felt and heard. About its centre was a rather sharp, irregular-edged gap. Some oedema of the upper lid was noticeable. Proptosis was very marked. Forwards the eye extended 13 mm., outwards 10 mm., and downwards 5 mm.

A history of severe influenza in the 1918 epidemic was given with several lighter subsequent attacks. On questioning as to injury he said that nine months previously he had a severe blow on the inner side of the left orbit through his head falling forward on to a tap, when recovering from gas in a dentist's chair. A bad " black eye " followed. About three months after this when blowing his nose a considerable quantity of thin yellowish fluid came away.

R.V.=6/6.

L.V.=6/36, with correction=6/12.

Muscles. Maddox rod, exophoria 2°. Hyperphoria 2°.
Maddox wing, exophoria 15°. Hyperphoria 0.5°.

Fundus normal.

Operation was advised, as condition was diagnosed as an ethmoidal mucocele.

A few days later the case was shown at a B.M.A. clinical meeting. The photograph here reproduced was taken March 1, 1928.

(Fig. 1.) Circumstances prevented the patient having the operation then, and I did not see him again till the following May 14. Condition then was much the same. Vision was a little poorer.

L.V.=6/36 barely. With correction=6/12.

He noticed that gentle finger pressure above in 11 o'clock position gave better vision. On testing this I found his statement true, and was able to improve vision to 6/12 without glasses.

FIG. 1.

I sent him to Mr. R. M. Leith, Port Elizabeth, who operated on May 22, and wrote to me that " it was a very large ethmoidal mucocele extending to the back of the orbit and floor of frontal sinus. The inner wall of the orbit was entirely absorbed . . . the displacement should right itself and it will be interesting to see if the vision improves."

June 25, 1928. Appearance much improved by operation: proptosis less.

L.V.=6/18. With correction=6/9.

August 13. Improving, but still some exophthalmos.

R.V.=6/4.5

L.V.=6/18. With correction=6/9. P.D.=6.2. Ord.

April 15, 1929. Very well. Glasses comfortable. No headaches. Left eye still a trifle anterior to right in position.

L.V.=6/18 improved to 6/5 $\frac{\text{cum} - 0.50\text{D.Sph.}}{\text{c.} + 2.25 \text{ Cyl. axis } 30° \text{ in.}}$

Hyperphoria 1°. Exophoria 1° (Maddox rod).
„ 2°. „ 14° (Maddox wing).

Fundus, normal. Photograph taken then reproduced. (Fig. 2.) Hyperphoria causes no discomfort: rides motor cycle.

August 15, 1929. The cylinder was reduced to +1.75 D. axis 30° in.

It is probable the diminished visual acuity was due to a certain

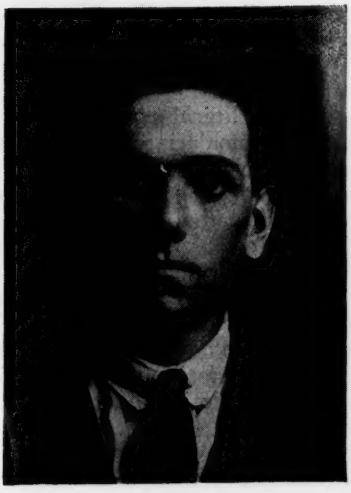

FIG. 2.

amount of retro-bulbar neuritis from distended ethmoid cells pressing on the optic nerve.

I am fortunate in having a record of the vision taken in April, 1922, aged 14½ years. He then came complaining of headaches and soreness of eyes. His vision was 6/9 in each eye with +1.0 D.Sph. +0.25D.Cyl. axis horizontal in R., and +1.0D.Sph. +0.5D.Cyl., axis horizontal, in left.

The diminution of his hypermetropia between 14 and 20 years is interesting and unusual. His previous correction was now much too strong. It does not accord with Duke-Elder's views: "In children hypermetropia tends normally to diminish with growth and consequently the refraction usually approaches emmetropia gradually until adolescence is passed."* My patient's hypermetropia continued to lessen much beyond that period.

* "The Practice of Refraction," Duke-Elder, p. 93.

AN INQUIRY INTO THE ORIGIN OF THE ABNORMAL PIGMENTATION OF THE SKIN AND CONJUNCTIVA IN CASES OF KERATOMALACIA IN ADULTS

BY

ARNOLD PILLAT

PROFESSOR OF OPHTHALMOLOGY, PEKING UNION MEDICAL COLLEGE

AND

GORDON KING

ASSOCIATE IN OBSTETRICS AND GYNAECOLOGY, PEKING UNION MEDICAL COLLEGE

ONE of the most striking clinical symptoms of xerosis epithelialis conjunctivae and of keratomalacia of adults in China is the pigmentation of the conjunctiva. In certain cases there is also a dark staining of the skin, though this is not always so easily recognizable. The pigmentation of the conjunctiva was visible in about 70-80 per cent. of all the cases of keratomalacia seen in Peking in the spring of 1929, but in varying intensity.

As far as the literature is obtainable, only in India and Japan has a similar pigmentation in cases of keratomalacia been reported. But there are considerable differences in the description of the pigmentation, as well as in the real nature of the pigment described, and there are nothing but theories about the origin of the pigmentation.

Wright,[2] in his paper on "Keratomalacia in Southern India," quotes the description of keratomalacia by Kirkpatrick,[1] who passes the pigmentation with the words (p. 168) "a well-marked icteric tinge is often now (*i.e.*, in advanced cases) added to the pigmentation of the conjunctiva." His description deals more or less with the keratomalacia of children. Wright,[2] speaking of the pigmentation seen in keratomalacia in adults, makes the following statement: (p. 171) "A very important, but inconstant feature is jaundice. Sometimes it is very marked and gives the scanty conjunctival débris in the fornix a greenish yellow colour. It appears to be more common in adults. It is sometimes associated with a small cirrhotic liver, at other times the liver is large. In one case a fairly well-to-do woman of middle age developed keratomalacia in association with an irregular, enlarged and painful liver, and intense jaundice probably of a malignant nature." And later in his paper he continues: "There are certain clinical observations which suggest that the liver plays a very important part in keratomalacia, but it cannot be assumed that this is more than secondary in its relation to the disease."

Contrary to Kirkpatrick and Wright, who seem to believe that

the pigmentation of the eye in keratomalacia is due to liver disturbances, Sh. Mori[3] states that the pigment is melanin pigment, because of its various reactions to different agents, but he does not give any explanation for the origin of the pigment. He says: "The explanation of the whole process of this kind of pigment formation is a very difficult thing to make, but I am at least sure that this melanin pigmentation comes as a result of xerosis and as a result of the epithelium cells losing their conjunctival nature because of abnormal dryness and coming to be like regular skin epithelium."

In the older literature the pigmentation of the conjunctiva is only mentioned by De Gouvea,[4] but no explanation is given.

Without going into further details, we would like to emphasize this peculiar dark brown colour of the pigmentation in keratomalacia, which has more the appearance of that seen in "argyrosis" or in Bronze-diabetes or in Addison's disease. The pigment itself seems to have the same hue in all cases. Even the slightest beginning of the pigmentation of the conjunctiva is as dark in its individual points as a very advanced case, only the greater number of the pigment dots explains the darker colour. The pigment lies chiefly in the basal cell layer of the epithelium and in the intercellular spaces, according to Mori's[3] and Pillat's[6] examinations. It advances from here into the middle layers and in severe cases extends almost to the surface of the kerato-hyalin layer of the thickened epithelium. The pigment increases in intensity as long as the keratomalacia progresses and disappears in the process of healing, but does so at a much slower rate than the other clinical symptoms.

Whether the discussion which has centred round the origin of the staining of the conjunctiva found in cases of "liver ophthalmia" (Baas, Weiss, Hori, Bamberger, Elschnig and Vollbracht[5]) has any bearing upon the origin of the pigment found in keratomalacia is doubtful. In keratomalacia the pigmentation is dark brown in colour whereas in cases of "liver ophthalmia" the colour is always described as "icteric" or "subicteric."

In order to determine what rôle the liver played in the production of this conjunctival pigmentation we undertook special liver function tests in a series of twelve cases of keratomalacia. The tests used were the laevulose tolerance test and the quantitative determination of the bilirubin in the blood serum. In order to facilitate the discovery of possible liver changes only severe and advanced cases of keratomalacia were chosen, no cases of xerosis epithelialis being included, although pigmentation is very often present in this condition.

The functions of the liver are so manifold that it is by no means easy to form a fair estimate of the efficiency of the organ by any

one test. The liver is mainly concerned with the metabolism of carbohydrates, proteins and fats, with the production of bile and with the excretion of various toxic substances from the blood stream into the alimentary canal. Clearly then liver efficiency tests may be based upon the ability of the organ to perform these various funtions. The laevulose tolerance test serves as an index of the metabolic capacity of the parenchyma cells of the liver. The estimation of the bilirubin in the blood serum gives an early indication of any accumulation of bile pigment. The eliminative power of the liver may be tested by the intravenous injection of certain substances such as phenol-tetrachlorphthalein : this dye is excreted solely by the liver and the rate at which it is removed from the circulation may serve as an index of liver function. For the purposes of the present investigation, the two first mentioned tests were chosen, as previous experience had demonstrated the clinical value of these methods[7] in determining the presence of pathological changes in the liver.

1. *The Laevulose Tolerance Test.*—The basis of this test is founded upon the remarkable power which the liver possesses for building up laevulose and storing it as glycogen. It has long been known that the administration of laevulose by the mouth produces practically no effect on the fasting blood sugar level. This is in marked contrast to glucose where a sharp rise in the blood sugar curve follows its ingestion. The reason has been shown to be that in the normal individual the laevulose is quickly absorbed from the intestine and passes through the portal system to the liver where it is rapidly polymerised and stored as glycogen with the result that little if any of the sugar ever reaches the systemic circulation and consequently there is little or no rise in the blood sugar curve. Very different results occur in cases where some pathological lesion of the liver cells exists. The diseased or injured cells are unable to carry out their function adequately and considerable quantities of the sugar escape their action and enter the systemic circulation, giving rise to a marked increase in the blood sugar level.

For practical purposes a patient is starved for eight hours and a dose of 50 gm. of laevulose is then given by mouth (the fasting blood sugar level having been determined immediately previously). Blood sugar estimations are then made half-hourly for two hours. The result of the test is plotted graphically and represents the laevulose tolerance curve. In normal cases the peak of the curve is reached in from half to one hour, the rise does not exceed the fasting level of blood sugar by more than 20 mg. per 100 c.c. of blood and the curve usually returns to the fasting level at the end of two hours. In abnormal cases the peak of the curve is delayed, the rise exceeds the fasting level by considerably more than 20 mg. per 100 c.c. and there is no return to the fasting level after two hours.

In the series of cases under discussion, with one exception, the laevulose tolerance curve yielded normal results. The accompanying Table shows the figures in detail. It will be seen that, excluding Case 4, the rise above the fasting level in mg. per 100 c.c. ranged between 3 mg. and 20 mg. with an average of 11 mg. Moreover, in practically every case the blood sugar curve approximates to the fasting level again at the end of the second hour. Fig. No. 1 shows quite a typical example of what may be regarded as a normal curve, found in Case No. 10.

TABLE SHOWING RESULTS OF LAEVULOSE TOLERANCE TEST AND SERUM BILIRUBIN ESTIMATIONS.

Case No.	Age	Diagnosis	Laevulose Tolerance Test Blood Sugar %					Bilirubin in blood serum
			Before Laevulose	1 hour later	1½ hours later	2 hours later	Rise in mg.	
1	24	Keratomalacia	0.110	0.117	0.117	0.114	7	Trace
2	24	,,	0.114	0.117	0.110	0.114	3	0.2 Unit
3	22	,,	0.096	0.094	0.101	0.094	5	0.8 Unit
4	23	,,	0.117	0.156	0.150	0.129	39	Trace
5	24	,,	0.091	0.107	0.104	0.104	16	0.8 Unit
6	25	,,	0.091	0.104	0.104	0.104	13	1.0 Unit
7	23	,,	0.101	0.110	0.107	0.107	9	Trace
8	19	,,	0.101	0.107	0.107	0.101	6	Trace
9	33	,,	0.114	0.129	0.125	0.117	15	Trace
10	21	,,	0.117	0.125	0.125	0.117	8	0.2 Unit
11	16	,,	0.114	0.134	0.131	0.121	20	Trace
12	21	,,	0.117	0.125	0.110	0.107	8	—

Case No. 4 was the only instance where an abnormally high curve was obtained. The patient had complained of symptoms for twenty days and on presenting himself at the hospital typical signs of keratomalacia were found in both eyes with pigmentation of the fornices. The skin showed numerous acneiform lesions with hyperpigmentation of the papules and scars. Physical examination showed no abnormality in the heart, lungs or abdomen. The

liver was not enlarged or tender. The urine was normal. The laevulose tolerance test, however, showed an abnormal result with a rise of 30 mg. above the fasting blood sugar level. A curve such as this would indicate a moderate degree of liver dysfunction—but it is to be noted that the accompanying bilirubin figure is normal. The significance of this curve, therefore, is not clear.

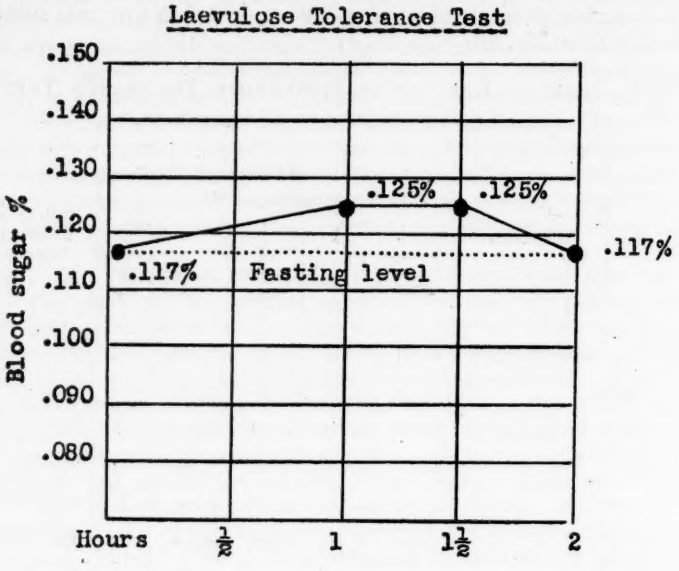

Fig. No. 1.- Normal Curve (see Case No. 10.)

With this one exception the laevulose tolerance test showed no evidence of impairment of the metabolic function of the liver in any of the other cases.

2. *Quantitative Estimation of Bilirubin in Blood-serum.*—One of the earliest and most constant results of disturbance of hepatic function is the accumulation of biliary constituents in the blood stream. In former years appreciation of this sign depended upon the clinical appearance of jaundice and upon the detection of bile in the urine. The important work of van den Bergh[8] and McNee[9, 10] has now rendered possible the estimation of such minute amounts of bilirubin as are present in the normal blood serum. Slight increases of bilirubin in the serum can be readily detected by means of the van den Bergh technique even when

jaundice is clinically inappreciable and the whole subject of " latent " jaundice has thus been given a fresh significance.

In the light of newer views it would appear that the polygonal liver cells are not essentially concerned with the manufacture of bile pigment but chiefly with the transfer of bile pigment from the blood capillaries to the capillaries and ducts of the bile system. The actual manufacture of bile pigment would seem to occur in the cells of the reticulo-endothelial system which are scattered throughout the body in the tissues of the liver, spleen, bone marrow, lymph glands and other organs. The cells of this system are concerned with the breaking down of haemoglobin and the elaboration of bile pigment. The bile pigment in its turn it set free into the blood stream and finally reaches the liver where it is transferred to the biliary ducts. It therefore follows that there is normally present in the blood stream a certain very small proportion of bile pigment. If the liver cells suffer any damage or functional derangement the proportion of bile pigment in the blood serum rises so that the detection of comparatively small increases may have a very important pathological significance.

van den Bergh introduced a technique for the quantitative estimation of these small amounts of bilirubin. He found that in normal blood serum bilirubin was present in quantities varying from 1 part in 1,000,000 to 1 part in 400,000. The estimation of such small quantities necessitated the fixation of an arbitrary standard and he therefore designated a strength of 1 part of bilirubin in 200,000 parts of serum as one unit. Normal serum therefore may contain anything from 0.2 to 0.5 units, rarely more. Further work demonstrated the fact that when the serum-bilirubin content rose to 4.0 units or higher bile appeared in the urine and jaundice became apparent clinically. Cases where the bilirubin content is higher than normal but less than 4.0 units are therefore to be regarded as instances of latent jaundice. The more severe degrees of jaundice would show anything from 20.0 to 50.0 units of bilirubin in the serum, and sometimes readings even higher than these are obtained.

A glance at the table given above will make it clear that no significantly high readings were obtained. In six of the cases only a trace of bilirubin was present which means that the concentration was below 0.2 unit. In two cases the low normal level of 0.2 unit was found.

In the remaining three cases in which the test was performed slightly higher readings were found (0.8 unit, 0.8 unit and 1.0 unit). A separate consideration of these cases may be justified.

Case No. 3.—The palpebral and bulbar conjunctivae of the right eye showed marked discoloration of a dirty brownish nature. Only slight discoloration was present in the left eye. The skin showed

many scabietic lesions with marked brownish hyperpigmentation. The heart and lungs were normal, but in the abdomen a tender area at the level of the umbilicus and to the right of the middle line was discovered. The liver and other organs were not palpable. The possibilities of cholecystitis or a liver abscess were borne in mind, but all abdominal symptoms disappeared in a couple of days and did not reappear. In this case the laevulose tolerance test was normal and there was 0.8 unit of bilirubin in blood serum.

Case No. 5.—Marked pigmentation of the conjunctivae was present, especially in the fornices. The colour resembled that found in argyrosis. The heart, lungs and abdomen were normal. The liver was not enlarged or tender. The laevulose tolerance test was normal and there was 0.8 unit of bilirubin in the serum.

Case No. 6.—There was slight greyish pigmentation of the conjunctivae. The heart, lungs and abdomen were normal, and the liver was neither enlarged nor tender. In this case, again, the laevulose tolerance was normal and there was 1.0 unit of bilirubin in the serum.

It will be seen that in each of these cases the bilirubin level was only slightly above the limits of normal, not approaching the 4.0 unit level at which jaundice ceases to be latent and becomes manifest clinically. It is doubtful whether so slight a rise would warrant the conclusion either that definite liver damage was present or that the conjunctival and cutaneous pigmentation owed their origin to an excess of bile pigment.

The present investigation, therefore, would seem to show that it is improbable that any marked degree of impairment of liver function is present in cases of keratomalacia and that the pigmentation found in such cases is probably not of biliary origin.

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ANNOTATIONS

Text Illustrations

In a journal such as ours text illustrations play a very important part. We willingly admit that our contributors have sometimes been justified in their complaints as to the quality of the reproduction, more especially of microphotographs. The Editorial Committee have devoted considerable thought to this question. There are obviously only two methods whereby an improvement could be effected. The first is the use of a more highly polished paper. The objections to this, however, would in our opinion outweigh the advantages. The glare from the surface and the added weight make any such journal uncomfortable to read. The second possibility is the greater use of plate paper. The objections to this are two: taking the illustrations away from the context renders reference to them less easy, and the binding of the volume is rendered more costly, as unless plates are properly " guarded " in, they soon part company from the volume; moreover the volume so made up would be much more bulky and weighty.

The Prevention of Blindness

An international association to aid in the prevention of blindness was constituted at the recent congress in Holland. The headquarters of the association will be in Paris, and the following have been chosen as the Executive Committee: President, Professor de Lapersonne, Paris; Vice-President, Dr. Park Lewis, United States; Secretary, Dr. Humbert; Treasurer, M. Demarchy; Members, Professor van der Hoeve (Holland), Professor de Grosz (Hungary), Professor Axenfeld (Germany), Professor Marquez (Spain), Professor Trantas (Greece), Mr. Bernard Cridland (England), Professor Maggiore (Italy), Dr. Isahara (Japan), and Dr. Szymanski (Poland). Two seats have been reserved for Central and South America.

ABSTRACTS

MISCELLANEOUS

(1) **Magnasco, M. (Rome).—On experimental tumours in the interior of the rat's eye. (Sui tumori sperimentale da innesto nell'ochio del ratto.)** *Saggi di Oftal.*, Vol. IV, 1929.

(1) **Magnasco** records in this paper the results of 110 ocular inoculations of rat-sarcoma in various parts of the eye. 31 of these inoculations were successful. His results were similar to those found by previous observers in other parts of the body, *i.e.*, they were more successful in young rats and in warm weather. In rats inoculated in both eyes simultaneously no unfavourable reciprocity was observed. Hypodermic and intravitreal injections on the same eye showed no influence on the intraocular tumour of the large hypodermic one. Inoculation between the layers of the cornea produced a remarkable growth which spread between the layers and also extra-corneally with secondary deposits in the anterior chamber. In general, however, the cornea, although readily infiltrated after inoculation of the anterior chamber, was not susceptible to direct inoculation. Although the anterior chamber has not proved a very good place for inoculation, the tumours tend to grow along the iris, shifting it either forwards or backwards or even completely replacing it. The vitreous appears to be a more suitable site of inoculation, the tumours developing to a great extent and involving the whole of the globe. The retina is very resistant to tumour infiltration, the retinal elements never being replaced by the neoplasm. The choroid becomes infiltrated with sarcoma cells, and the lens becomes opaque. Retrobulbar inoculations are also usually successful, but the optic nerve is never invaded and the sclera is highly resistant. The paper is illustrated by numerous microphotographs which are reproduced with great beauty.

E. E. H.

(2) **Siegfried, W. (Zürich).—Experimental research on the supposed injurious effect of ultra-violet rays on the adaptation of the eye. (Experimentelle Untersuchungen ueber den angeblich schadigenden Einfluss der ultra-violetten Strahlen auf die Adaptation des Auges.)** *Arch. f. Ophthal.*, Vol. CXX, 1928.

(2) Disturbance of adaptation has recently been added to the list of diseases of the eye that have been attributed by writers to the action of ultra-violet rays, and it was to test this point that

Siegfried conducted a series of experiments, in which he studied the effect of the highly concentrated ultra-violet rays from a mercury-vapour lamp and the light of an ordinary electric bulb.

It was found that with the ultra-violet lamp there is no appreciable diminution of adaptation even after 15 minutes' action on the macular area, while the light from an electric bulb, filtered and not further concentrated, reduces it considerably.

He will not admit the objection that in these experiments the action of the ultra-violet rays was very short and temporary, while the results from chronic exposure to the rays might be perhaps reversed: the concentration of these rays and the length of exposure, he holds, were too great, and moreover the electric bulb, from which the ultra-violet rays had been almost completely eliminated, produced a marked diminution. The slight change caused by the mercury-lamp was probably mainly a fluorescence effect.

This research confirms previous work by Vogt on the same subject.

THOS. SNOWBALL.

(3) **Aubaret, E. and Sédan, Jean (Marseilles).—"Angor Ocularis."** *La Clin. Ophtal.*, September, 1928.

(3) "Angor ocularis," or in French "angoise oculaire" appears to be a general term for ocular angiospasm and particularly for spasm of the retinal arteries. **Aubaret** and **Sédan** after a short discussion of the mechanism of vascular spasm, in which they are unable to enlighten us very much on account of its extreme complexity, say that they themselves prefer to reserve the term "Angor ocularis" for a certain "*catégorie de faits,*" and the particular type which they have in mind is that in which the symptoms are analogous to those of angina pectoris. Whereas in the latter trouble the outstanding symptom is the imminence of cardiac stoppage, so in the former there is the sensation of the sudden loss of vision which provokes the feeling of anguish or despair. The sudden, more or less complete blindness, lasting for minutes or hours, is very rarely accompanied by frontal or orbital headache and is thus to be distinguished from ophthalmic migraine with which it is apt to be confused. Again, "The essential characters which distinguish angor ocularis from other forms of angiospasm, particularly those of cortical origin, are the following: unilaterality is very common whereas bilaterality is constant in cortical cases; the blindness is incomplete as compared with the absolute blindness in cortical cases." The authors regard the condition of angor ocularis as a primary affection and distinguish other forms which are secondary to, *e.g.*, chronic glaucoma or retinal arteriosclerosis.

These secondary types they distinguish as "*pétite angoise oculaire.*" The authors ask "Do the subjects of repeated attacks of angor ocularis run the risk of developing definite retinal lesions?" They hold that Horniker in 1927 has established the fact that they do, in the long run, develop such lesions which consist of fine haemorrhagic and exudative lines at the posterior pole and especially at the macular region. The authors have something to say about immediate treatment by such drugs as amyl nitrite, nitrate of soda, trinitrine, etc., but their final paragraph is perhaps of more importance for it gives the advice to look carefully for the cause, especially an endogenous or exogenous intoxication, or, not to be forgotten, syphilis, either hereditary or acquired.

ERNEST THOMSON.

(4) **Wilmer, W. H. (Baltimore, Md.).—Clinical aspects of ocular tuberculosis.** *Arch. of Ophthal.*, January, 1928.

(4) **Wilmer's** paper opens with a brief historical survey of this condition in which he refers to Meller's statement in 1926 that tuberculosis is a very much more important cause of irido-cyclitis than is generally thought. In ordinary cases there can be no certainty of the exact locality of the lesion, but practical proof of the nature of the infection can be obtained by therapeutic use of tuberculin which, in Meller's clinic, is used in every case of irido-cyclitis. Löwenstein says much the same in connection with choroiditis which is only rarely associated with active pulmonary tuberculosis. Wilmer's series of cases comprises those which have usually passed the early and acute stages of disease in which the eyes have been already severely damaged and where the disease has proved itself resistant to ordinary treatment. In all the cases he surveys syphilis and focal infections have been excluded as possible causes. The percentage of cases in which a tuberculous aetiology was revealed is roughly 40. The diagnosis was made by the occurrence of a high degree of hypersensitivity to tuberculin. The cases may be grouped as follows :—

(1) Uveitis with mutton-fat keratic precipitates, sometimes synechiae, pupillary exudate (in severe cases), chorio-retinitis, disseminated or limited to the macular region. In this type there may or may not be other tuberculous foci. Two cases are quoted, both showing improvement under tuberculin therapy.

(2) The inflammation is confined to the posterior segment of the uvea. Vitreous opacities, if present, are fine and dustlike. There is usually a large yellowish white area of exudation, with haemorrhages and older, slightly pigmented, spots round it. In the later stages there is massive fibrosis. These cases require very careful

treatment with small doses of tuberculin; one is quoted in the text.

(3) Cases of recurrent haemorrhage into the vitreous in youthful patients, with subsequent retinitis proliferans, if untreated. The total number of these cases is not given; in all but one, however, the cause was proved to be tubercle.

The technique used by the author for diagnosis was: to give an intradermal injection of 1/10,000 mg., if this was negative 1/100 mg. was used and then 1/10 mg. For therapeutic purposes, the injections were made subcutaneously starting with 1/1,000,000 mg. of B.E. or O.T. and working the dose up gradually to 1 mg., or even more, according to whether there was any reaction. In his conclusions, the author suggests that the milder cases of ocular tuberculosis may be due to a strain of bacilli less virulent than those which cause phthisis but of sufficient virulence to cause a general tuberculous condition evidenced by pasty complexion, clammy extremities, low blood pressure, etc. As the occurrence of tuberculosis in the eye renders it more vulnerable to the attacks of other organisms, foci of sepsis should be carefully searched for and eradicated when found.

F. A. WILLIAMSON-NOBLE.

(5) **Derby, George S. and Carvill, Maud (Boston).—Anterior ocular tuberculosis.** *Arch. of Ophthal.*, November, 1927.

(5) **Derby and Carvill's** cases comprise a group of 130 patients of which they were able to locate 63. The group included a large number of cases of severe phlyctenular disease, often associated with scleritis or sclerosing keratitis. No patient was under observation for a period of less than 10 years, and some had been seen for 20 years. The authors give an interesting summary of their work and the following are some of their conclusions:—Tuberculosis of the eye is a chronic disease which may be overcome for a time, but it is extremely likely to recur if the patient's general resistance be lowered. Nearly 10 per cent. of the cases died from phthisis though none of them showed active lesions in the lungs when first seen, and all were living a careful life with regard to general hygiene, diet, etc. The average attack of anterior ocular tuberculosis is self-limited and recovery may occur in two to three months or not for a year. The length of the remission depends on the general health of the patient. The disease has, up to the present been regarded too lightly and it should be classed with the arrested case of pulmonary tuberculosis. Unless the resistance is maintained at a high level these patients are liable to a succession of local recurrences and to development of serious tuberculosis.

F. A. WILLIAMSON-NOBLE.

(6) **Berhausen, Oscar (Cincinnati, Ohio).—Tuberculin therapy in ocular tuberculosis.** *Arch. of Ophthal.*, November, 1928.

(6) **Berhausen** recognises three stages in the development of tuberculosis. The primary consists in proliferation of the fixed connective tissue cells, with the production of epithelioid cells, and the subsequent production of giant cells and changes in the surrounding tissue which tend to isolate the tubercle. The second stage is that of generalisation of the infection, *via* the blood or lymph streams, with appearance of foci in distant parts—and is similar to a chronic bacteriaemia with anaphylactic symptoms. The tertiary stage is that in which immunity has been reached and the inflammatory process is less marked. In the eye these stages are exemplified as follows:—the first by a small nodule in the iris with a slight area of resorption, the second by diffuse iritis with acute toxic symptoms, and the third by torpid iritis with formation of glass-like nodules and precipitates. Cases in the second stage are best treated by rest and general measures, specific medication being reserved for those in the third stage. The commonest position for the primary lesion in ocular tuberculosis is in the peribronchial lymph glands, and 80 per cent. of cases show X-ray findings in the chest; a thorough physical examination is therefore imperative. With regard to diagnostic tests, the author seems to favour the stick reaction in which a drop of tuberculin is placed on the sterilised arm, and a sterile darning needle is used to introduce the tuberculin intracutaneously. Short histories are given of 17 cases treated with tuberculin, 10 of which showed both local and general improvement, and five a small degree of improvement. The patients who gave the best results were those who reacted in a general way by elevation of temperature and showed also some local reaction in the eye. Most of them were treated with B.E. or T.R.

F. A. WILLIAMSON-NOBLE.

BOOK NOTICES

The History and Traditions of the Moorfields Eye Hospital. One Hundred Years of Ophthalmic Discovery and Development. By E. TREACHER COLLINS. With 27 plates, Pp. 226. London: H. K. Lewis and Co. 1929. Price, 12s. 6d. net.

This admirable account of the history and traditions of Moorfields Eye Hospital has laid all ophthalmologists under a debt of gratitude to the author. No one is better qualified to carry out the

task than Mr. Treacher Collins. His long association with the hospital as clinical assistant, junior and senior house surgeon, curator of the Museum, surgeon, and consulting surgeon, and finally as member of the managing committee has endowed him with unsurpassed knowledge and experience. These alone would not suffice to ensure success in the undertaking. That could be attained only by laborious delving into minute books, reports, and biographies, and then too only when inspired by pious love and respect for what is in the deepest sense the author's Alma Mater.

The history of "Moorfields" must naturally appeal most strongly to past and present "Moorfields men." And who are Moorfields men? There is scarcely an English-speaking ophthalmologist who is not in some sense a Moorfields man. There are those most nearly associated with the hospital as members of the staff and clinical assistants. Of the latter group many have gone forth into various parts of the British Isles and British Empire and have carried Moorfields teachings and traditions with them. The missionary work began early in the history of the hospital, for only two or three years after its foundation in 1804 by Saunders, his pupil, William Adams, founded the West of England Eye Infirmary at Exeter, which is still a flourishing institution. Both of these men were pupils of a surgeon of Barnstaple, named John Hill, who "was bold enough in those pre-anaesthetic days to operate for cataract. It was from him also that Saunders first learnt the value of the use of belladonna for dilatation of the pupil." It would seem that this obscure country practitioner inspired the founder of Moorfields with an interest in ophthalmology, and it is satisfactory that his name has been rescued from oblivion.

Later on in 1819, Mr. R. Richardson, who had studied at Moorfields under Benjamin Travers, founded the Madras Eye Infirmary, which still exists as the Government Ophthalmic Hospital, Madras.

The missionary work was not limited to the British Empire. Moorfields attracted students from the United States of America. Among them in the early days were Dr. Edward Delafield and Dr. J. Kearney Rodgers, who on their return to New York founded the New York Eye and Ear Infirmary in 1820. Another American student was Dr. Edward Reynolds, who, on his return to Boston, founded the Massachusetts Charitable Eye and Ear Infirmary in 1824. Both these hospitals are still flourishing.

Thus was the fame of Moorfields spread far and wide, and from then onwards up to the present day, it has been the Mecca of ophthalmologists from all parts of the world. It is said to be more blessed to give than to receive, and Moorfields has been lavish in its gifts. But it is due to that very fact that the bread cast upon the waters has come back to it day after day. For to these re-visits of

old friends, and visits of strangers and foreigners is largely due the stimulus not only to keep abreast of the times, but to forge ahead and retain the lead. May it ever be so!

Moorfields was founded in 1804, but was not opened for the reception of patients till March, 1805. A few months before an Eye Dispensary was opened in Cork Street, so that Moorfields was not literally the first ophthalmic hospital opened. The Cork Street establishment was not very successful and was closed down in 1872, the balance of its funds (£200) being handed over to Moorfields.

The hospital was first called the London Dispensary for Curing Diseases of the Eye and Ear. In 1808 the treatment of ear diseases was given up. It started at No. 40 Charterhouse Square. In 1821 it was removed to Moorfields, near Finsbury Circus, and was re-christened the London Ophthalmic Infirmary. The name was again changed in 1837 to the Royal London Ophthalmic Hospital, and it was removed to City Road, its present site, in 1899. Thus, Moorfields Eye Hospital, a name known throughout the world and the favourite designation given to it by its alumni, has never been its official title.

From its earliest days, Moorfields has been in the forefront of advance in clinical ophthalmic medicine and surgery. Saunders was a pioneer in the needling of cataract in children, and Saunders's needle, still used, was named after him. The medical side was not neglected, as is shown by the appointment of Dr. Farre as physician in 1805.

The Moorfields medical school is almost coeval with the Hospital, for in 1811, on the recommendation of Dr. Farre and Mr. Travers the practice of the Infirmary was opened to medical students and permission was granted to the medical officers to deliver lectures. It was a surgeon of the hospital, Sir William Lawrence, who first advocated that a course of instruction in diseases of the eye should be included in the medical curriculum. Research received a great impetus with the arrival of Sir William Bowman, a great physiologist and ophthalmologist, a member of the great triumvirate, Bowman, Graefe and Donders. The development of the medical school owes much to Marcus Gunn, who instituted more regular courses and more systematic teaching. From this nucleus has grown the elaborate curriculum which is now carried out at the hospital.

In 1828 Dr. Farre started a short-lived Journal, which may be regarded as the forerunner of the "Royal London Ophthalmic Hospital Reports," which commenced in 1857. Thus the first English journal devoted specially to ophthalmology originated with the staff of Moorfields Hospital. Many distinguished men, such as Bowman, William Mackenzie, Hughlings Jackson, Argyll

Robertson, Jonathan Hutchinson, Nettleship, contributed papers to the Reports, which may be justly said to contain the best original work done in ophthalmology in England during their publication. The Reports were finally absorbed with the *Ophthalmic Review* and the *Ophthalmoscope*, in this Journal in 1916.

To Moorfields must also be ascribed the honour of the foundation of the Ophthalmological Society of the United Kingdom in 1880.

But why attempt further to epitomise a history which is so fascinatingly written in Mr. Treacher Collins's book? Our object is attained if we succeed in directing our readers' attention to the pleasure in store for them when they read it. It is well written, well printed, and well illustrated with portraits of Moorfields' worthies. We have found but few misprints or mistakes: but for the sake of future editions which will certainly be called for, we would point out that there is no "Walleston" Medal of the Royal Society (p. 112—probably the Wollaston Medal of the Geological Society is meant), that Canada is not a " colony " (p.142), and that Streatfeild is probably turning in his grave at finding his name written " Streatfield " throughout the volume.

Squint. By CLAUD WORTH. Pp. 246, 42 illustrations. Baillière, Tindall and Cox. 1929. Price, 10s. 6d.

The author is to be congratulated upon the appearance of the sixth edition of his well-known and classical work and upon the fact that there has been no need to make any fundamental alterations in his book since its original appearance twenty-six years ago.

The present edition therefore differs very little from the preceding one. The main additions are a plea for a more thorough training of young surgeons in the technique of squint operations, and a statement that the author, in private work, is in the habit of inserting seven to nine very fine sutures in an advancement operation, instead of using the two knotted sutures formerly described. This makes the operation more complicated and one is doubtful whether it can be performed so satisfactorily by surgeons who lack the author's long experience and knowledge of how much to advance the muscle in order to correct a given deformity.

Catalogue of Lewis's Medical and Scientific Circulating Library. London: H. K. LEWIS and CO., LTD. Pp. 576. 1928. Price, 15s. (to subscribers, 7s. 6d.)

The present edition of this useful catalogue has been revised and brought up to date to the end of the year 1927. It is arranged in two sections; Part I, Authors and Titles. Part II, Classified Index of Subjects, with the names of authors who have written upon them.

In Part I can be seen at a glance the date of publication, the published price and the size of the book, while Part II is a model of lucid compression. The whole must have entailed a great amount of work, and the proprietors of the Library are to be congratulated upon the result, which forms a most useful book of reference.

IN MEMORIAM

EDWARD ERSKINE HENDERSON, M.B., F.R.C.S.

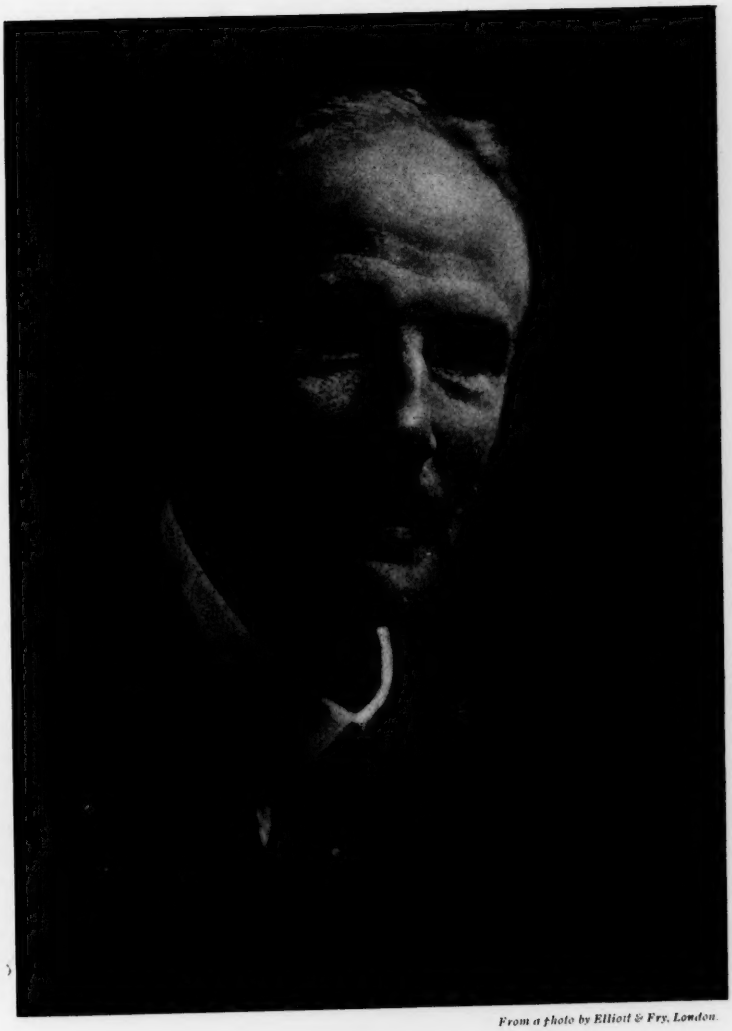
Quis desiderio sit pudor aut modus
Tam cari capitis?

"WHAT restraint or limit should there be to grief for one so dear?"

The news of the sudden death of Edward Erskine Henderson, on August 23, came as a great shock to his many friends and is an especially heavy blow to THE BRITISH JOURNAL OF OPHTHALMOLOGY.

The eldest son of the late Edward Henderson, M.D., of Shanghai, Henderson was born in China in February, 1870. His father was a scion of an ancient Scottish family, of whose kinship to Sir Walter Scott, Edward Erskine Henderson was proud. The father went to practise in Shanghai in the sixties of last century; and in after years the firm of Henderson, McLeod and Jennings Milles, the junior partner having been at one time house surgeon at Moorfields, became one of the best known firms in the East.

Edward was sent home to England to be educated and entered at Cheltenham; even at this early age his deafness was manifest and it was decided to remove him from Cheltenham and enter him at Harrow. At Harrow he did well and reached the sixth form. His classical education here was of the greatest advantage in enabling him to render important service to British ophthalmology in editing this journal for the past six years. From Harrow he went to St. John's College, Cambridge, and thence to Guy's Hospital. Having qualified at Cambridge in 1895, he held house office at Guy's, as house surgeon to Mr. Golding Bird and obstetric assistant to Dr. Peter Horrocks. He married on leaving the hospital and went out to join his father's practice, where he held the appointments of assistant surgeon to the Shanghai Hospital and to the police force.



From a photo by Elliott & Fry, London.

EDWARD ERSKINE HENDERSON

1870—1929.

Henderson returned to England in 1898 to work for the F.R.C.S., and began the study of ophthalmology at Moorfields in the clinics of Sir John Tweedy and Mr. William Lang. Later he worked with Mr. Lang and with Mr. Lawford. He went through the mill and was a candidate for the post of curator of the museum at the election when Coats was successful; he never concealed his opinion that in electing Coats, the hospital secured the best man. At Moorfields Henderson worked for the long period of fifteen years. As a clinician he was above the average while his manipulative dexterity made him a first class operator; and yet his early promise was never fulfilled: his deafness handicapped him in the race for clinical appointments and beyond holding the ophthalmic surgeonship to the West Ham Hospital, and for a very short period, early in his career, the assistant surgeonship to out-patients and pathologist at the Royal Eye Hospital, he achieved no high position in the hospital world. He retired from practice in 1920. For twenty years, however, he held a unique position in the English ophthalmic world as editor, first of the *Ophthalmic Review* and then of this Journal. He took over the editorship of the *Ophthalmic Review* in 1910 and steered it through a very critical time until it was amalgamated with the *Royal London Ophthalmic Hospital Reports* and the *Ophthalmoscope* in THE BRITISH JOURNAL OF OPHTHALMOLOGY. He was appointed sub-editor to Mr. Sydney Stephenson and on the latter's death in 1923, he became senior editor. Those of us who have worked with him month by month during the latter years know how great a service he performed for British ophthalmology. He will be greatly missed; his critical mind, his shrewd judgment and his sense of humour made him an ideal colleague. He took the greatest interest in the journal; he improved our English and our punctuation and sat in kindly judgment on our faults. With Professor Starling he was the author of an important piece of research on the factors which determine the production of the intraocular fluid; this work was published in the *Journal of Physiology* in 1904, and in the *Proceedings of the Royal Society* in 1906, but apart from this and the routine editorial work he wrote little. With the late R. H. Clarke he did a great deal of work on the minute anatomy of the central nervous system; in this work Henderson was responsible for all the photographic reproductions, and this was the work on which he himself set most value. An expert proof reader he was much in demand among his friends for correcting the proofs of their works.

No more stimulating teacher than Henderson could be imagined; he had a knack of rubbing in the essentials of ophthalmology that may not have been exactly relished at the time, but which was bound to stick in the memory, and his generosity was unbounded.

Outside his work he had many interests; in his younger days, a fine horseman, he was also a good shot, especially with the pistol, and he competed regularly at Bisley for some years before the war. He played a good game of golf and he was on a fishing holiday in Ireland when he died. He was well read, with an especial affection for the works of R. L. Stevenson; he was expert in heraldic matters and he did beautiful work in wood and ivory on the lathe, in which he was entirely self taught. But no catalogue of his excellencies can fill the gap caused by his death, and to the writer, who has known him intimately for more than twenty years, the blank is irreparable. In the words of Cicero,—*Moveor enim tali amico orbatus, qualis, ut arbitror, nemo unquam erit, ut confirmare possum, nemo certe fuit.* "For I am indeed moved by the loss of a friend such, I believe, as I shall never have again, and—as I can assert on positive knowledge—a friend such as no other man ever was to me."

Henderson's eldest son was killed in the Great War in 1918; to his widow and his surviving children the sympathy of all ophthalmic surgeons will go out in their bereavement.

Mr. Lawford writes:—

"As one who for many years was closely associated with E. E. Henderson in hospital and editorial work, I should like to add a few words expressive of my high regard for him and of my admiration for his character. Henderson's clinical work was always on a high level and his careful help and co-operation in the Outpatient room at Moorfields was such as to evoke my lasting gratitude.

It was a great satisfaction to me personally when he consented, with some reluctance, to join the BRITISH JOURNAL OF OPHTHALMOLOGY as one of the Editors. I felt certain that his services to the Journal (then being launched) would prove of great value: that feeling has been more than realised.

His untimely death has diminished my circle of trusty colleagues and has robbed me of a valued friend."

NOTES

The Wilmer Ophthalmological Institute

EXERCISES for the dedication of the Wilmer Ophthalmological Institute will be held on October 15 and 16, 1929, under the auspices of the Johns Hopkins University, the Johns Hopkins Hospital and the William Holland Wilmer Foundation. An appropriate programme has been arranged including the following addresses: "The Development of Ophthalmology in Europe," by Hofrath Ernst Fuchs, of the University of Vienna; "Some Contributions and Phases of American Ophthalmology," by Dr. George E. de Schweinitz, of the University of Pennsylvania; and "Colour Vision and its Anomalies," by Sir John Herbert Parsons, F.R.S., of the University of London.

* * * *

North of England Ophthalmological Society

A SERIES of three lectures will be delivered under the auspices of this Society by Professor van der Hoeve, in January next, as follows:—

January 17, "X-ray diagnosis in ophthalmology," at Sheffield.
January 20, "Strabismus," at Leeds.
January 21, "Tumours and pseudo-tumours of the eye and orbit," at Manchester.

Members of the medical profession interested in ophthalmology who wish to hear these lectures will be welcomed by the Society. Details of time and place will be announced later.

* * * *

The International Trachoma Prize

THE Royal Hungarian Minister of Public Welfare and Labour offers a prize of 2,000 Swiss francs as a reward for an independent work on the subject of the aetiology of trachoma. The work in question must signify valuable progress in this sphere.

Essays intended for the competition must be addressed to the Eye Clinic No. 1 of the Royal Hungarian Peter Pázmány University, Budapest (VIII, Mária-utca 39) not later than June 30, 1931.

Works that have already appeared in print are eligible for this competition, as well as works specially written for the occasion. The Jury of Award may also consider works not submitted for competition but appearing in print between July 1, 1929, and June 30,

1931. The essays submitted may be written in German, English, French, Italian or Hungarian.

The Jury of Award will be appointed by the Royal Hungarian Minister of Public Welfare and Labour, and its decision will be announced not later than December 31, 1931.

By order of the Royal Hungarian Minister of Public Welfare:

Prof. EMILE DE GROSZ, M.D.

* * * *

Reading without Spectacles in 1695

IN the diary* of Abraham de la Pryme, the Yorkshire antiquary, in the year 1695, occurs the following note, "There was at Bramwith near Hatfield an old clerk that could scarce ever get a pair of spectacles that he could see with, his sight was either so vitiated or destroyed. At last an old wife tells him a way how he might see without spectacles—to get a prayer book printed upon yellow paper. At last he got such a one, and tho' it was but a small print, yet I observed that (he) saw and read with as much ease as if it had been ever so bigg." We trust that the printing of this note will not lead either to a revolution in the printing industry or to a complete cessation of the work of the opticians.

FUTURE ARRANGEMENTS

1929

October 11.—Section of Ophthalmology, Royal Society of Medicine.

October 18.—North of England Ophthalmological Society, at Manchester.

November 8.—Section of Ophthalmology, Royal Society of Medicine.

November 15.—North of England Ophthalmological Society, at Bradford.

December 6.—North of England Ophthalmological Society, at Leeds.

December 13.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting.)

*Surtees Society, Vol. LIV.

1930

January 10.—Section of Ophthalmology, Royal Society of Medicine.

January 17.—North of England Ophthalmological Society, address by Professor Van der Hoeve at Sheffield. Subject: "X-ray Diagnosis in Ophthalmology."

January 20.—North of England Ophthalmological Society, address by Professor Van der Hoeve at Leeds. Subject: "Strabismus."

January 21.—North of England Ophthalmological Society, address by Professor Van der Hoeve at Manchester. Subject: "Tumours and Pseudo-Tumours of the Eye and Orbit."

February 7.—North of England Ophthalmological Society, at Newcastle-on-Tyne.

February 14.—Section of Ophthalmology, Royal Society of Medicine.

March 7.—North of England Ophthalmological Society, at Liverpool.

March 14.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting.)

March 28.—North of England Ophthalmological Society, at Sheffield.

June 13.—Section of Ophthalmology, Royal Society of Medicine. (Annual Meeting.)

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *August, 1929.*

SCARLETT. Circinate retinitis; report of a case.
BENGTSON. The epithelial cell inclusions of trachoma; experimental studies.
FINNOFF and THYGESON. The finding of bacterium granulosis (Noguchi) in trachoma.
MARTIN, W. O., Junr. Anterior and posterior dialysis of the retina.
COWAN and MARCOVE. A method of mapping scotomata with tangent screen by binocular fixation.
FERREE, RAND and MONROE. Studies in perimetry (3). Errors of refraction, age, and sex in relation to size of the form field.

Annales d'Oculistique. *July, 1929.*

MÉCHITCH. Experiments on the rational construction and the ophthalmological and surgical application of giant electro-magnets.
MAGITOT. The symptomatology and the problem of pathogenesis of glaucoma.
ARRUGA. The bevelling of the bony openings in the operations for the treatment of lacrimation.

Archives d'Ophtalmologie. *August, 1929.*

COPPEZ. On pseudo-foreign bodies in the eye.
STROHL. Perception of relief and stereoscopic acuity.
DOUMER. Osmotic drainage in ophthalmology.
DEJEAN. Notes on the form and structure of the vitreous body with the slit-lamp.
TRANTAS. Senile lesions of the anterior capsule of the lens and of the pupillary margin.
NOISZEWSKI. Connections between the pressure of the cerebro-spinal fluid and of the intraocular fluid.
RAGAIN. Treatment of trachoma by high frequency

Zeitschrift für Augenheilkunde. *August, 1929.*

HAMANN. Observations on the diagnosis and treatment of tumours of the hypophysis.
PRESSBURGER. On cystic degeneration of the periphery of the retina.
SATTLER. Apparatus for objective and subjective measurement of the angle of squints with graphic recording of the field of fixation and investigation of the field of vision according to Bjerrum.
GUTZEIT. On familial nodular keratitis.
BARCZINSKI. A case of ophthalmomyiasis interna anterior.
FAZAKAS. On a new method of Denig's transplantation treatment for pannus.
UMINISSA CHANUM MUSSA-BEILI. A case of paralysis of the abducens after influenza.

Bollettino d'Oculistica. *August, 1929.*

GUGLIANETTI. Contribution to the histopathology of chronic catarrhal dacryocystitis with particular regard to the plasma cells.
ACCARDI. Xanthomatosis bulbi—experimental researches.
GALLENGA. Contribution to our knowledge of the veins of the naso-lacrimal canal.
MARIOTTI. Tuberculous or lymphogranulomatous choroiditis ?
DE CRISTOFARO. Detachment of the retina in a glaucomatous patient.

Archivos de Oftalmologia Hispano-Americanos. *August, 1929.*

LUQUE. Vascular changes in glaucoma patients.
ESCUDERO. Experiences with radium therapy in spring catarrh.
NEVOT. Ultra-violet light baths in the treatment of so-called lymphatic kerato-conjunctivitis.
BASTERRA. Late results of dacryocysto-rhinostomy.
DOMINGUEZ. On a case of primary glaucoma with matutinal hypotonia.

Revista Cubana de Oftalmologia y Oto-Rino-Laringologia.

July, 1929.

ESTÉBAN. On so-called hole at the macula.
FINLAY. Modern conceptions as to the nature and origin of the intraocular fluids and their influence on the ocular tension.